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Effects and mechanisms of action of nitric oxide on transmitter release in mouse motor nerve terminals

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Abstract

We examined the mechanisms of NO action on transmitter release in neuromuscular preparations of the mouse diaphragm muscle using a standard microelectrode technique. A donor of NO (SNAP, 100 μM) and a substrate for NO synthesis (L-arginine, 100 μM) reduced the evoked transmitter release from motor nerve endings. At the same time, SNAP did not change the frequency and amplitude of miniature end-plate potentials, while an inhibitor of NO synthase, L-NAME, exerted no effect on evoked and spontaneous transmitter release. Inhibition of soluble guanylate cyclase by ODQ (2.5 μM) abolished the effect of NO on the evoked transmitter release, while the elevation of cGMP-level by its membranepenetrating analog 8BrcGMP did not prevent such effect. The elevation of intracellular concentration of cAMP by 100 μM of its analog 8(4CPT) cAMP or inhibition of phosphodiesterase (PDE) by the action of 100 μM IBMX eliminated NO effects on transmitter release. It is concluded that NO activates soluble guanylate cyclase and intensifies the cGMP synthesis. Activation of the PDE II via an increase in the cGMP level with consequent reduction of the level of intracellular cAMP and decrease in the activity of PKA reduced transmitter release from mouse motor nerve ending. © 2012 Springer Science+Business Media New York.

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Keywords

cAMP, cGMP, guanylyl cyclase, neuromuscular junction, nitric oxide